

### Regarding “Differential gene expression in human abdominal aorta: Aneurysmal versus occlusive disease”

In the article by Armstrong et al,<sup>1</sup> the expression of 16 MMPs was evaluated in both abdominal aortic aneurysm (AAA) and arterial occlusive disease (AOD) specimens. In this paper, the MMP-9 results overexpressed in both tissues. The remaining MMPs revealed strikingly similar expression. Recently Carrel et al<sup>2</sup> demonstrated that MMP-3 was overexpressed in AAA tissue samples and may be involved in aneurysm pathogenesis. These papers prompted us to report our data on MMP-3 genotype in the two groups of patients.

It is well known that the insertion/deletion mechanism of an adenosine nucleotide (A) at -1171 bp in the MMP-3 gene promoter sequence results in a polymorphism (5A/6A) in which the transcriptional activity of the 5A homozygous is approximately double that of the 6A homozygotes.<sup>3</sup>

We genotyped for 5A/6A polymorphism on MMP-3 promoter 58 patients with AAA, 57 with AOD, and 133 age-matched and sex-matched healthy controls. All subjects were Italians.

The genotype distribution (AOD, 3 5A/5A, 27 5A/6A, 27 6A/6A; AAA, 10 5A/5A, 35 5A/6A, 13 6A/6A; Controls, 36 5A/5A, 59 5A/6A, 38 6A/6A) was significantly different among AOD patients and both AAA patients ( $P = .007$ ;  $\chi^2$ , 9.69) and controls ( $P = .001$ ;  $\chi^2$ , 13.44). No differences were observed between AAA patients and controls ( $P = .11$ ).

In AOD patients, the less expressive allele 6A was significantly more represented than the wild type 5A (AOD, 0.71; versus AAA, 0.52;  $P = .0039$ ; and versus controls, 0.51;  $P = .0002$ ).

Our results confirm the Finnish ones that showed no MMP-3 genotype differences in AAA patients<sup>4</sup> but, for the first time, evidenced a significant high 6A frequency in AOD patients, in whom atherosclerosis manifests by narrowing of the arterial lumen. These results are consistent with the finding that 6A allele is significantly more represented in subjects suffering from internal carotid artery stenosis, another occlusive arterial disease.<sup>5</sup> On this basis, some evidence appears that studies should be carried out to verify the hypothesis that the MMP-3 promoter polymorphism 5A/6A might be involved in AOD pathogenesis as a genetic marker or new risk factor.

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doi:10.1067/mva.2003.44